## On Books

Activity Anorexia: A Review of Solving the Anorexia Puzzle: A Scientific Approach by W. F. Epling and W. D. Pierce

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Anorexia nervosa is the name of an eating disorder in which people, mostly young women, starve themselves to a severe, sometimes fatal, level. Solving the Anorexia Puzzle: A Scientific Approach, written by two behavior analysts, is a unique treatment of what causes and maintains this condition. The authors emphasize, and support with much of their own laboratory data, the interactions among biology, conditioned behavior, and environmental influences in the development and maintenance of this syndrome.

The book is divided into three major parts (Anorexia Nervosa; Activity Anorexia; Clinical Aspects of Activity Anorexia) with 12 chapters and an index. There is a foreword by P. J. V. Beumont, a researcher and clinician in Australia, and an introduction by the authors. Each chapter begins with a list of main topics and a helpful summary and has its own reference list with citations referred to by number in the text. These features make the book very appropriate for any course that deals with eating disorders. Part 1 is a review of the discovery of anorexia and presents the findings that traditionally describe and identify the syndrome. There are data of incidence, populations at risk, and recovery statistics. Death rate estimates vary from 3% to 25%. The authors show that traditional treatments, such as psychoanalysis, insight psychotherapy, family therapy, and cognitive psychotherapy typically produce no better re-

Toronto: Hogrefe & Huber Publishers, 1991. Reprints can be obtained from Carl Cheney, Department of Psychology, Utah State University, Logan,

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sults than the 66% of individuals who recover without treatment! Therefore, any therapy must top the 66% spontaneous recovery rate to be considered effective.

In Part 2, the authors continue to review the medical and psychological literature concerning anorexia nervosa and simultaneously begin to weave their own commentary and critique into the narrative. They analyze the condition not as a psychological or purely biological disorder but as a complex phenomenon resulting from a combination of biological, behavioral, and environmental factors. Their central theme in Part 2 is that some of the cases currently labeled "anorexia nervosa" under DSM-III-R (perhaps as many as 75%) are actually better characterized in terms of a distinct, new biobehavioral syndrome, which they call "activity anorexia."

The authors have conceived this new syndrome to label the behavior generated when reduction in appetite and excessive exercise occur together. Much of the evidence for this new syndrome comes from the authors' own research with rodents. For example, a critical finding on which the authors depend heavily is that when rats are given one meal per day and simultaneous access to a running wheel, the rats will literally run themselves to death (Routtenberg & Kuznesof, 1967). One might expect animals to adapt to food shortage by decreasing activity and conserving energy. However, as discussed below, that is not what appears to be biologically programmed to occur.

Epling and Pierce have thoroughly replicated and investigated this phenomenon. They and their students have researched the question of what could possibly be the reinforcer for such seemingly maladaptive (in the laboratory) behavior. Abundant evidence indicates that a restricted food intake regimen generates activity in rats and apparently in humans as well, even though such behavior appears to violate common sense. The increase in activity produces the release of an intrinsic reinforcer in the brain, which the authors persuasively argue to be beta endorphins. During prolonged exercise the brain actually produces its own reinforcer (e.g., runner's high). This biological set of circumstances, together with the social pressures to be thin and the dieting that develops, are what the authors contend support excessive selfstarvation behavior, or what they call activity anorexia, in humans.

The activity-based anorexia theory follows nicely from what is known about the neurophysiological origins of feeding behavior (e.g., Kupferman, 1985). Organisms have evolved internal mechanisms that monitor nutrient availability. When levels are low, the motor system is activated. Active animals increase the probability of encountering food; obviously, such a mechanism has survival value. The organism also has a built-in system by which to distinguish good food from bad (Booth, 1984). Starting with these physiological processes (nutrient monitoring, motor activation, consequence discrimination), one can proceed to show how more and more complex behaviors can be established. The fooddeprived active organism operates on the environment and produces consequences that increase or decrease the probability of such response classes in the future. Repertoires are thus selected from the interaction of biological processes and environmental contingencies. That is to say that animals are built to behave when nutrient levels decline. This general behaving then allows for the environment to select repertoires differentially by means of the application of consequences (Cheney, 1991).

The authors provide an ongoing primer of operant conditioning as they explain stimulus control, response topographies, and dependent measures, as well as the

effects and side-effects of behavior consequences. This feature of the book helps clarify the authors' analytical orientation and assists the reader in grasping their position. Because this analysis of anorexia stems from the position that behavior—even this seemingly maladaptive behavior-is a function of its consequences, Epling and Pierce are forced to locate functional antecedent and consequent stimuli to fit a behavior-analytic account. This they do very well in terms of describing the social pressures to be thin that our culture generates, which, in turn, lead to dieting, and then produce an increase in activity, the combination of which spirals into a condition of critical starvation. Each of these elements in the activity anorexia cycle is treated as a component of a three-term contingency, including the biological substrate for the appetite-exercise connection. Aspects of the biological component are speculated to have an evolutionary basis when individuals who moved when food became scarce were reinforced for such behavior and were therefore selected for survival by the environment. If such behavior had a genetic component, then this component would likely become widespread.

The oft-repeated thesis of the book is that when rats or humans are food deprived (e.g., by being restricted to only one meal a day) they, by virtue of their physiology, become more active. Physical activity then acts to devalue food as a reinforcer and simultaneously produces the release of brain opiates (the beta endorphins) that intrinsically reinforce activity. The combination of food restriction and physical activity produces a "multiplier effect" that generates more activity and less eating. This is the fundamental basis of activity anorexia.

The authors describe the major medical and psychoanalytic models of anorexia nervosa to which they contrast behavioral, cultural, and their own biobehavioral model. Traditional (psychological) assessment procedures, treatments, outcomes, and prognoses following therapy are described and discussed at length and compared with the authors'

analysis. In treating this topic, Epling and Pierce employ an ecological orientation, in the sense that they develop the evolutionary, biological, and environmental survival aspects of activity anorexia, which is unlike any of the alternative accounts. That is, their biobehavioral model of activity anorexia is a completely new classification of what they consider to be a separate form of anorexia nervosa. Most accounts of anorexia include the observation of increased activity as part of the syndrome, but Epling and Pierce appear to be the first to propose such behavior as the major contributor to the problem. Their theory makes a great deal of sense, and it is stated in such a way that it can be tested in part or as a unified syndrome.

In Part 3 of their book, the authors distinguish between the classification of anorexia (nervosa or activity) as a personality disorder and one based on functional relationships. They then show how a functional analysis contributes to effective treatment recommendations as well as to a better understanding of the problem than other approaches provide.

Case studies from the literature are reviewed and include a description of traditional medical and psychological assessments and treatments. The case studies were selected to provide support for each component of the authors' theory in one way or another, yet they appear to be representative of the syndrome and its treatment. Epling and Pierce suggest that the evaluation of any treatment should be considered simply in terms of whether the patient recovers from selfstarvation and remains at appropriate weight. Psychodynamic treatments often fail to correct activity anorexia because anorexia is considered to be a mental illness. When hypothetical internal "psychological" states are assumed to be the source of the syndrome, therapy is often misdirected and inappropriate. Psychological symptoms (e.g., depression) can arise after the development of weight loss (anorexia) and excessive exercise, but they do not precede or initiate the problem.

The increased activity spurred by restricted food availability makes good evolutionary survival sense for the organism, especially for rats. By becoming active in the face of a food shortage, an animal is more likely to discover food somewhere in its environment. Furthermore, because a single presentation of food is not sufficient for long-term survival, one large meal a day actually induces even more activity. This aspect of the activity anorexia account sounds vaguely reminiscent of the old straightalley study in which, following stable running times, a meal is placed half-way down the alley, yet the rat, instead of stopping and eating, continues on to the goal-box. Activity anorexia might also contain features of a behavior contrast phenomenon, in which reduced reinforcement in one context leads to increased activity in another. Heightened activity causes the release of opiate-like substances in the brain; this has been shown to suppress appetite when exercise is intensive or when body weight is low.

The book focuses on one anorexia symptom at a time and elaborates the importance of each from the perspective of research with nonhumans. The book is an excellent example of how to organize and analyze a large and diverse corpus of data according to a unifying theoretical principle, while presenting the components of an applied analysis of behavior.

The authors do not report the actual use of their own recommended therapy, which follows from the biobehavioral activity theory. This is not a failure of the book, however, because Epling and Pierce did not set out to illustrate a therapy. Instead, their goal is to show how basic research has led to a plausible and well-reasoned data-based statement as to what are probably the controlling variables of this serious behavioral pathology. I believe this treatise to be a very important breakthrough in discovering and describing the controlling variables of this disorder.

For the reader who is sophisticated in behavioral principles, selected chapters may be skimmed, as there is some redundancy in coverage. However, this very feature makes the book appealing as an educational and persuasive text. The theme is repeated, and many of the same reference citations occur frequently. Aside from these minor concerns, the book is a scholarly and novel treatment of this troublesome eating disorder.

## **REFERENCES**

Booth, D. A. (1984). Food-conditioned eating preferences and aversions with interoceptive elements: Conditioned appetites and satieties. In N. Braveman & P. Bronstein (Eds.), Experimental assessments and clinical applications of con-

ditioned food aversions (pp. 22-41). New York: Annals of the New York Academy of Science.

Cheney, C. D. (1991). The source and control of behavior. In W. Ishaq (Ed.), *Human behavior in today's world* (pp. 73-86). New York: Praeger.

Kupferman, I. (1985). Hypothalamus and limbic system II: Motivation. In E. Kandel & J. Schwartz (Eds.), *Principles of neural science* (2nd ed., pp. 626-635). New York: Elsevier.

Routtenberg, A., & Kuznesof, A. W. (1967). "Self-starvation" of rats living in activity wheels on a restricted feeding schedule. *Journal of Comparative and Physiological Psychology*, 64, 414-421.